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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Delayed Effects of Seemingly Minor Head Injuries

BENNO SCHLESINGER, M.D.

New York, New York

Whenever a head injury gives rise to local signs that appear after a free interval, an epidural hemorrhage or a subcortical clot may be developing. The clinical picture of epidural hemorrhage may further be simulated by a cerebrospinal fluid accumulation in the subdural space or, rarely, by fat embolism. Temporary minor improvement may also follow the original shock caused by contusions.

We propose to divide the available material into two groups. Group A comprises cases in which post-traumatic aphasia of delayed onset is a prominent sign. In group B are cases in which various other organic disturbances occur after a free interval. In two of the seven cases the injuries were fatal. They will serve to illustrate the fact that the serious effects of some cranio-cerebral injuries do not necessarily reflect the force of the trauma, as expressed in physical terms and that severe damage to the brain may be the result of an apparently insignficant injury.

GROUP A

Case I:

On January 27, 1944, B. S., a 60 year old man, fell from a height of about seven feet striking the left side of his face and head. He was unconscious for a short time but was able to return home unassisted. Several hours later the patient became nauseated and vomited. The family physician examined him at 3 p.m. and found no neurologic signs. At 6 p.m. the patient was less responsive; he was examined by a neurologist who found a right lower facial weakness. The blood pressure was 155/110. At 11 p.m., when I saw the patient, he appeared to be somewhat drowsy. In addition to the facial weakness there was now weakness in the right upper exterimty. The abdominal reflexes were absent on the right. The right pupil reacted to light. The left could not be seen because of marked swelling of the eyelid on that side. The pulse rate was 72. The clinical examination was otherwise negative.

Comment:

Here was a patient who had sustained what appeared to be a mild concussion, but who soon afterward developed a right lower facial weakness and weakness in the ipsilateral upper extremity. Drowsiness also appeared after a free interval. Although the diagnosis of a post-traumatic epidural hemorrhage was considered, surgical interference seemed unwarranted because signs of increased intracranial pressure (if any) were out of proportion to the focal signs.

Subsequent Course:

1) Five days after the accident the patient showed complete motor aphasia and a right lower facial weakness which, however, was present on volitional innervation only. The weakness in the right upper extremity had progressed to almost complete paralysis while motor power in the right lower extremity was unimpaired. Roent-

From the Bronx Hospital, New York, N. Y.

genologic examination of the skull revealed a slightly depressed fracture in the left posterior parietal region measuring three centimeters in diameter. The mild clouding of consciousness noted on the evening following the accident was no longer present.

Surgical intervention for suspected epidural clot was again declined in view of the discrepancy between the progressive local and the subsiding general signs. As the right lower extremity was not involved, a subcortical hemorrhage of any size also seemed unlikely. The site of the fracture did not correspond to the part of the brain responsible for the clinical picture whatever the pathology of the injury might have been. Elevation of the fracture was suggested to the family, but consent to operation was refused.

2) Three weeks after the accident the patient was still aphasic, although to a somewhat lesser degree. On the other hand, the previously uninvolved right lower extremity had now become paretic. The weakness, however, was not marked enough to prevent the patient from walking without assistance, while the paresis in the right upper extremity remained complete.

In spite of the recent onset of weakness in the lower extremity indicating progression of the pathological process a subcortical clot seemed to be unlikely as the early signs revealed a slight but definite tendency to improve. The clinical diagnosis was cumulative softening.

The patient's condition became stabilized and he left the hospital about two weeks after the last neurological examination. Due to the pressure of work at that time he was lost sight of and there were no follow up studies. A spinal tap had not been performed.

Case II:

On March 7, 1949, at 9 a.m., J. Å., a 49 year old man fell from a height of about six feet resulting in a fracture of his left forearm and multiple abrasions on the left side of his face. The patient who had been unconscious for a few minutes following the accident was able to return home after first aid treatment by the local physician. In the evening the patient's wife noted that he was unable to talk and that he had some difficulty in swallowing.

When I saw the patient at 4 p.m. on the day following the accident he had a masticatory seizure, the jaw being drawn to the right at regular intervals. There was no concomitant vocalization and no irritative phenomena in other parts of the body were noted. The patient was fully conscious during the attack.

Examination immediately following the seizure revealed a sharp deviation of the outstretched tongue to the right, and a relatively mild lower facial weakness on the same side. There was complete motor asphasia, agraphia and alexia, reading being tested by writing simple orders. As far as could be determined reading appeared to be less impaired than speaking and writing as the patient immediately noted whether a newspaper page was handed to him upside down or in a correct fashion. (There were no pictures on this page that might have guided the patient.) There was some apraxia of the face and tongue, as evidenced by his futile efforts to protrude his tongue on command and the concomitant distortion of the facial muscles. At other times, however, he was able to protrude his tongue, although not in the midline. Otherwise the neurologic examination was completely negative. In particular, there were no motor or reflex changes in the right upper extremity and the abdominal reflexes were equal and active on both sides.

Following the seizure the patient was unable to void for several hours. The temperature rose to 101 F, and there was mild tachycardia. There was no nuchal rigidity and no neurological cause for the elevated temperature could be found. (No spinal tap was done.) The clinical examination was otherwise negative. X-ray examination of the skull and blood Wa R yielded normal findings.

Comment:

While the motor aphasia, agraphia and alexia as well as weakness in the right side of the tongue and lower face developed after a free interval following the head injury operative intervention appeared to be unwarranted because of the discrepancy be-

tween the massive motor aphasia and the absence of signs of abnormalities in the right upper extremity and of intracranial hypertension.

Subsequent Course:

Four days after the accident the patient made sustained and partly successful efforts to talk and write. Writing appeared to be less severely impaired in that the patient occasionally succeeded in printing a word correctly. The partial ability to read and write was episodic, however, and whatever short utterances the patient was able to produce were made in a propulsive, abrupt manner. On one occasion he was able to read aloud a short sentence written by the examiner. Otherwise the neurologic findings were unchanged. The temperature was normal.

Two weeks after the accident the patient was able to speak one or two sentences but inflection of speech was still conspicuously altered. His ability to read and to write was improved.* The neurological findings were unchanged and temperature was normal.

Four weeks after the accident the patient's ability to talk, read and write was almost completely restored. The following is an excerpt from the protocol.

"Whenever I talk too long I get mixed up whatever it is." Do you find it difficult to pronounce certain words? "Yes, for instance, music. If I do not think of it, it comes out like moosic, like in the old country, but when I think of it, it comes out all right. If I read too fast, I cannot soak it in, I would not know what I was reading about, my mind is a little behind. Whenever I put the pencil on paper I could not put down of whatever I think. In my mind I knew what the doctor's name was but I could not communicate with him and no way of telling what I wanted. I could not get the last name of the boys which were in my business, just the first name."—Have you any Hungarian friends with whom you speak in Hungarian? Yes, but they seem to speak English all the time, we never speak Hungarian. (The patient had been in the United States about 20 years.) As far as could be determined the inflection of speech was normal. There were no difficulties in articulation. Making allowance for the fact the patient had learned to speak English at a relatively late age, his ability to express himself was satisfactory.

The neurologic examination yielded essentially the same findings as before. The hypoglossal palsy was perhaps slightly less noticeable and the facial weakness less conspicuous.

Discussion

In both our cases of mild concussion following a trauma to the left side of the head motor aphasia, alexia and agraphia and motor signs were delayed in onset. Clearly, the findings cannot be attributed to the concussion but to some associated pathological process. Motor aphasia combined with motor symptoms occurs either in massive lesions of the left premotor region, which do not necessarily involve Broca's center, or in small lesions of the posterior portion of the inferior frontal convolution. According to Davison, Goodhart and Needles[2] motor aphasia combined with hemiplegia may likewise develop incident to rupture of the lenticulostriate artery. As a massive lesion could be excluded on clinical grounds and a complete hemiplegia failed to develop in either case it is reasonable to assume that the head injury had given rise to a relatively small focus particularly in case II. Since the onset of aphasia was not immediate, as in Nielsen's[3] case II, a cortical contusion can be excluded. Furthermore, the disturbance subsided in the first patient while his right lower extremity became involved as late as three weeks after the accident.

Apart from the history, the clinical course of the disease was similar to that observed in cases of spontaneous softening due to arteriosclerosis. A comparison with three such cases reported by Lévy,[4] in which motor aphasia associated with motor signs developed without antecedent head injury shows a remarkable similarity between the traumatic and the non-traumatic group (Table I) and we are therefore inclined to believe that the underlying pathological process was a gradually developing thrombosis of a local artery supplying the cortex and subcortex in the region of Broca's center.

Autopsy in Lévy's first case revealed an elongated cortical softening involving the

*In spite of his inability to make himself easily understood and despite the injury to his arm he was quite cheerful and very anxious to overcome his speech difficulties. He tried to talk to his roommates and practiced eagerly with pencil and paper. Certainly the speech defect could not be attributed in this case to any form of embarrassment and feeling of insecurity resulting from the brain lesion as such, as recently claimed by Alford[1] for agnostic, aphasic and similar disorders.

hind end of the second and completely destroying the posterior part of the third frontal convolutions. It extended as far as the island of Reil, the precentral gyrus and the ependyma of the lateral ventricle. In the second case the softening involved the walls of the central fissure and extended as far as the triangular portion of the third frontal convolution. The lenticular body was intact but the malacia reached deeply into the white matter of the frontal lobe. There were multiple small softenings involving the junction between the two inferior frontal convolutions on the one hand and the precentral gyrus on the other in the third case.

Before attempting to reconstruct the sequence of events in our cases it may be pointed out that Broca's center is supplied by at least two arteries, which have been given different names by various observers, but similarly identified by all. Lévy, whose description is the simplest and most logical, recognizes a prerolandic and an external orbito-frontal artery. The former supplies approximately the inferior two thirds of the precentral gyrus and the adjacent part of the second and third frontal convolution; the latter, the second and third frontal convolution with the exception of their anterior segments which are supplied by the anterior cerebral artery. Both the precentral and external orbito-frontal artery participate in varying proportion in the supply of the operculum of the third frontal gyrus, anastomoses between the two vessels taking place in the pia mater. Broca's center has therefore a composite blood supply.

As is well known the ramifications of the Sylvian artery were meticulously studied by French writers, especially Moutier,[5] Tixier[6] and Lévy.[4] Moutier, in his monumental work "L'aphasie de Broca" expounded the view that the region in question is supplied by a recurrent vessel which accounts for its vulnerability. This opinion, however, is not shared by either Tixier or Lévy, in fact, the former stresses the ample blood supply of what is known in the French literature as "le cap." It is not without interest in this connection that Corning[7] published a picture of the hemisphere of an embryo upon whose surface the recurrent course of the vessel in question can already be recognized above and in front of the still open Sylvian groove. As in other cortical territories the local ramifications of the Sylvian artery are evidently subject to many variations.

We are inclined to believe that in our first case softening of the prerolandic artery produced a partial hemiplegia which was followed by motor aphasia. The speech disturbance may be attributed to the extension of the thrombosis into the arterial branch supplying Broca's center. At the same time, the patency of the main vessel became still further reduced and the weakness in the right upper extremity increased to complete paralysis. Three weeks after the accident the patient developed weakness in his right lower extremity due to progressive softening in the upper precentral gyrus which depends for its blood supply not only on the anterior cerebral but also on its anastomosis with the prerolandic artery. At the same time, the aphasia showed a tendency to improve. Whether this was due to improved collateral circulation through the external orbito-frontal artery or some other mechanism such as vicarious functioning of the minor hemisphere is not immediately apparent. Collateral supply of the leg area by the anterior cerebral artery, while insufficient to maintain an adequate circulation, prevented the paresis in the right lower extremity from becoming complete. Weakness in the ipsilateral lower extremity was likewise present in two out of the three non-traumatic cases reported by Lévy.

In our second case, the motor paralysis involved the cortical foci of the 7th and 12th cranial nerves on the right. There was but insignificant improvement of the hypoglossal and facial weakness four weeks after the injury. Thrombosis probably took place in a small branch of the prerolandic artery which supplies the lowermost portion of the precentral gyrus. The motor aphasia which was markedly improved after two weeks had completely subsided after one month. As it would be difficult to reconcile this rapid improvement with a lesion involving Broca's center directly, one would conclude that the disturbance of speech developed as a result

of circulatory embarrassment in the adjacent lower precentral gyrus rather than in the hind end of the third frontal convolution. In other words, the posterior part of the rolandic operculum may have been involved only indirectly and recovery aided by collateral blood supply via the external orbito-frontal artery.

The dependence of brain function on vascular supply may perhaps be formulated in the following way: (a) complete loss of function is due to breakdown of the local circulation, (b) partial loss of function (either permanent or very prolonged) to inadequate vascular supply, and (c) transitory loss of function of short duration (either partial or complete) to derangement of the circulation in adjacent vascular territories, which has repercussions on the nutrition of the primarily uninvolved area.

Taking into account the great vulnerability of the neurons of the central nervous system re-establishment of the circulation should have played a relatively insignificant role. On the other hand, the rapid recovery in our second case can hardly be accounted for by the vicarious function of the sound hemisphere, considered by some to be the most important auxiliary mechanism governing restitution of function. Most likely recovery was aided by collateral circulation.

Case III

The third case, (Rawak's[8] case 2) may be briefly reported in the light of the pathophysiologic considerations outlined above.

The patient, a 43 year old man, while at work struck the left side of his forehead against a wood beam. He became dizzy and had to abandon his work but felt well enough to attend a meeting later in the day. Two days after the injury he suddenly developed motor aphasia, agraphia and weakness in his right upper extremity. Within a few months after the accident the aphasia was hardly noticeable; two years later it reappeared without apparent cause and became complicated by disorders of expressive speech. At the same time, the motor manifestations became associated with ipsilateral hemihypaesthesia and marked disturbances in intellect, affect and behaviour. Pneumoencephalography revealed enlargement of the anterior horn of the left lateral ventricle and great irregularity of its anterolateral outline on the side of the injury, displacement of the entire supra tentorial ventricular system toward the side of the lesion, and increase of air over the left frontal pole.

The author discarded the possibility that the disorder could have been caused by a non-traumatic condition and believed that the injury had given rise to encephalomalacia followed by brain atrophy. He did not comment on the possible mechanism of the vascular damage which he believed had occurred in this case.

There can scarcely be any doubt that the pathological changes were not confined to the vascular areas which were involved in our first two cases (I and II), but involved also branches of the anterior cerebral artery and eventually spread into the rolandic and posterior temporal arteries.

GROUP B

Case I

W.G., a 30 year old man, was injured 4/21/47 when a car he was driving collided with a truck. The patient remembered that he heard a crashing sound and that he found himself lying across the driver's seat when he regained consciousness. The window on his side was cracked and the car was turned around. He was dazed after the accident but did not vomit. Although the patient must have been thrown against the window pane there was no external evidence of injury to his head.

On the following day, while riding in a train, the patient experienced what he described as a "strange feeling in his brain" accompanied by severe headaches. About six weeks after the accident he began having frequent left frontal headaches and dizzy spells, the former coming on spontaneously or on change of position. Quick movements of his head almost invariably produced severe throbbing headaches in the left frontal region. The dizzy spells consisted in a "flashing" feeling, following which everything seemed to reel and were accompanied by the appearance of pin point black spots in front of his eyes. During

these spells, the patient would have a feeling of uncertainty and would have to hold on to something for support. For a few moments he was not sure about the position of his head and body. Upon questioning he reported that people who witnessed these spells saw him sway and stagger. In addition to these dizzy spells the patient experienced a delusion that the objects around him suddenly started to move. The seizures were regularly followed by headaches and, lately, by nausea. Following an interval of relatively well being and practical disappearance of what were in all likelihood kinaesthetic hallucinations as may occur in vestibular seizures the above described episodes returned and the patient became increasingly irritable and unsociable.

On neurologic examination right lower facial weakness and a sinking tendency (latent paresis) in the left upper extremity were found. The tendon reflexes, however, were more active in the left lower and right upper extremity.

Pneumoencephalography performed six months after the injury showed abnormalities of both the ventricles and the subarachnoid spaces. The AP views showed atrophy of both frontal lobes as evidenced by marked enlargement of the cortical channels and a hydrocephalus of the frontal horn. The difference in the size of the ventricles was striking. Lateral views revealed the extension of the process in the sagittal diameter of the skull, the alterations being visible in the posterior half of the frontal lobe, the rolandic region and the rostral portion of the parietal lobe. The atrophy appeared to involve predominantly the first frontal convolution on either side, chiefly on the right, where it extends into the subcortex. The degree of the atrophy is reflected by the size of the shadow of the rostral portion of the corpus callosum, which is thinned out. The territory involved corresponds roughly to the irrigation area of external frontal branches of the anterior cerebral arteries.*

Fig. 1. Case II, Group B. Pneumoencephalogram, AP view. Note marked thinning of the corpus callosum.

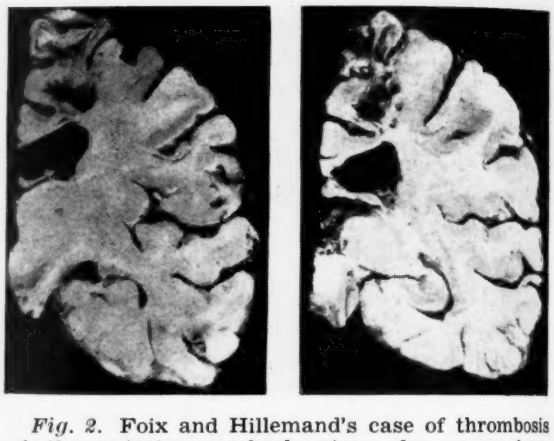

Fig. 2. Foix and Hillemand's case of thrombosis of the anterior cerebral artery, for comparison. (L'Enecephale, 1920, 20: p. 26.)

Case II

A 53 year old man, (Rawak,[8] case 1) slipped and fell, striking the left side of his forehead on the ground. He was unconscious for a moment but able to get up and walk home unassisted. He developed headaches and vomiting, which reappeared on the following day and were accompanied by dizziness on change of head position. At times the attacks of dizziness would come on spontaneously and appeared to be associated with momentary clouding of consciousness. The patient gradually became apprehensive and very sensitive to noise, increasingly forgetful and found it difficult to

*Unfortunately, the films are not available, but the pneumoencephalograms resemble, in all essential points, those reproduced in Fig. 1.

concentrate. He eventually lost all initiative and would sit around for hours at a time without moving or talking. It may be of importance to note that he was not entitled to any compensation benefits. The neurologic examination was negative except for head tremor which was continuous and of very fine amplitude.

Pneumoencephalography demonstrated dilatation of the left frontal horn and enlarged cortical fluid spaces overlying the left frontal pole. The anterior segment of the corpus callosum was thinned out. (Fig. 1.)

Comment:

The author does not express an opinion as to the mechanism of the brain atrophy but merely mentions the possibility that the condition might be due to softening or multiple small hemorrhages. Except for the fact that the cortical atrophy is less accentuated the pneumoencephalogram resembles the one in the previous case. For this reason, traumatic damage to the branches of the anterior cerebral artery supplying the genu and rostrum of the corpus callosum seems most likely.

Case III

A woman aged 20 (Couville and Olsen,[9] case 1) developed a left hemiparesis and hemihypaesthesia following a minor concussion. The patient died 13 days after the injury, (three days after right fronto parietal craniotomy for suspected interval intradural hemorrhage). At autopsy, a softening corresponding to the area supplied by the right middle cerebral artery was found.

Case IV

A man aged 48 years, (Courville and Olsen,[9] case 2), sustained a mild concussion as a result of a trauma to the right frontal region. He was unable to continue his work after a couple of hours. Three days after the accident he developed headaches, drowsiness and convulsions. He died five and one half days after the injury. At autopsy, recent thrombi in the proximal portion of both the right anterior and middle cerebral arteries were found which had produced extensive cortical and subcortical softening of the right cerebral hemisphere.

Comment:

Courville and Olsen feel that the encephalomalacia found in their cases can be accounted for by the thrombus in the regional vessels. At the same time, they express the opinion that atheromatous changes can be ruled out and that trauma was the only predisposing and exciting cause.

It is not unreasonable to assume that the vascular changes produced by severe head injuries may provide a clue as to the nature of the changes referable to a minor trauma. However, this does not appear to be the case here.

In a case reported by de Veer and Browder[10] the patient had fallen from a great height, landed on his head and was unconscious and motionless for a period of 20 minutes. The lucid interval lasted 12 hours. At operation, carried out on the assumption that an interval hemorrhage had developed, discoloration of the temporal convolutions was seen. At autopsy the media of the middle cerebral artery was found to be torn, denuded and fragmented. Shreds of media were identified in the clot which had become propagated into the internal carotid artery.

Since the vessel wall in Courville and Olsen's case 2 had been found normal (examination of the vessel at the site of thrombus formation having been omitted in their first case), the similarity of the effects of the injury is not reflected in the anatomical findings, and the mechanism of thrombus formation may therefore be entirely different.

Conclusions

The cases of craniocerebral trauma forming the basis of this report have the following clinical features in common: The initial effect of the injury is a mild concussion followed by a free interval ranging from several hours to several days. The free interval is terminated by the rather acute onset of various neurologic disturbances indicating focal damage to the brain and referable to the implication of either the anterior or the middle cerebral artery or both. In the two cases of Courville and Olsen, the lesion was identified at autopsy as encephalomalacia

and, in one of them, a thrombus occluding the middle cerebral artery was found. In the two cases reported by Rawak, and in one of our own three cases, both external and internal atrophy of the brain close to the site of the trauma was encountered, the correlation being particularly striking in Rawak's two cases. In the two remaining cases the pathological process was neither verified at autopsy nor visualized by air studies but the clinical picture corresponded to that seen in spontaneous arterial thrombosis on an atheromatous basis. Furthermore it could be demonstrated that the distribution of the lesions was not haphazard, but corresponded to mutually related arterial territories. Although conclusive proof is lacking we are inclined to believe that more or less extensive softening occurred in all seven cases.

Once the clinical picture is fully developed it can be readily differentiated from the various interval syndromes as well as the postconcussion syndrome although it may include elements of both. During the state of its evolution, however, "interval softening" may give rise to diagnostic difficulties. Some of them have been mentioned in the comment on cases I and II, Group A, and the reasons for correlating the various clinical signs with the involvement of the various arteries and their branches have been given in some detail.

While the diagnosis of posttraumatic cerebral thrombosis in our three personally observed cases rests on circumstantial evidence only, we have indicated why we felt justified in excluding a subcortical hemorrhage. We wish to point out that posttraumatic intracerebral clots give rise to progressive clinical manifestations and that deepening coma, signs of increased intracranial pressure and ispilateral enlargement of the pupil associated with other pupillary abnormalities are present either singly or in combination.[11,12,13,14] The progressive course of the disease (with or without preceding lucid interval) is probably the most important clinical sign[15] and confusion of the condition with epidural clots is therefore common, as has been known for a long time.[16] In our cases, on the other hand, there was either noticeable improvement in some spheres of function, accompanied by deterioration in others over a period of weeks or generalized improvement until such time as the patient's condition became stabilized.

Furthermore, the encephalogram in case I, Group B, showed bilateral frontal lobe atrophy duplicating, for all practical purposes the pathologic features in a case of thrombosis of the anterior cerebral artery. (Fig. 1.) Clinically the case under discussion could hardly be considered as bilateral frontal lobe hemorrhage neither could the bilateral hydrocephalus of the anterior horn and the atrophy of the frontal cortex be explained by the presence of either cysts or scars which replace the clots after varying periods of time.

It is hardly necessary to emphasize that vascular damage occurs only in a very small percentage of cases that sustain minor craniocerebral injuries. Since the site of the lesion appears to be grossly related to the vascular area involved one might infer that

(a) regional changes in that part of the brain which bears the brunt of the blow may produce abrupt changes in the physiologic state of the arterial walls, perhaps a temporary spasm or vasoparalysis which, in turn, may lead to thrombus formation in predisposed individuals, and that

(b) the appearance of the various symptoms and signs after a free interval coincides with a critical stage of the growth of the thrombus.

The fact that the trauma was a minor one in every instance and therefore failed to produce immediate local destruction may be of significance in that interference with the function of the local vasoreceptors, as a prerequisite for reflex changes, did not occur. We have, however, no explanation to offer as to the individual predisposition which we assume to represent the ultimate etiologic factor of thrombus formation. It may be conjectured that the cases described in this article represent borderline conditions in which the vascular changes were particularly severe while in the majority of instances widely scattered alterations occur-

ring in terminal ramifications rather than in major arterial branches would produce cortical palings or other minor changes. They may or may not give rise to neurological abnormalities and to roentgenologically demonstrable atrophic changes.

While the pathophysiologic mechanism invoked for the phenomenon of motor aphasia of delayed onset, and related neurologic signs is conjectural, and the concept developed should be regarded, for the present time, as a working hypothesis, the occurrence of serious disturbances following seemingly trivial trauma cannot be doubted. The rigid and apparently plausible correlation between the force of the trauma on the one hand, and the severity of the pathophysiologic effects on the other, which appears to be taken for granted by some should be discouraged. Conversely, the sequelae of severe craniocerebral injuries may eventually turn out to be minimal, and the concept of what constitutes a "mild" and what should be regarded as a "severe" head injury should therefore be revised and more sharply defined.

Summary

1) Three cases of minor craniocerebral injury are reported; in two motor aphasia of delayed onset was a prominent feature.

2) These cases were correlated, clinically and pathologically, with instances of apparently mild concussion, followed either by delayed focal signs and death, or by neurologic and pneumoencephalographic evidence of damage to the brain.

3) The possible pathogenesis of the cerebral changes is discussed.

4) Attention is called to the potentially misleading term "minor cranio-cerebral injury."

TABLE I

	Lower facial weakness	Hypoglossal weakness	Weakness in upper extremity	Weakness in lower extremity	Motor aphasia	Alexia	Agraphia
Author's case I (Group A)	+		+	slight	+	+	+
Author's case II (Group A)	+	+			+	+	+
Lèvy's case 1 (Observ. XXXII)					+	+	+
Lèvy's case 2 (Observ. XXXIII)					+	+	+
Lèvy's case 3 (Observ. XXXIV	+	+	+	slight	+	+	....
	....	+	+	slight	+	+	+

Note: The mild amnestic aphasic disturbances in our case II are of purely theoretical interest and have therefore not been indicated in the table, nor considered in the discussion.

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Investigations Into the Dynamics of Paranoid Reactions

(The Mechanism of Transvaluation)

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We owe to Freud insight into a psychological mechanism to which he gave the name "projection." This mechanism of defense against anxiety attributes to others, one's own character traits, attitudes, motives, or desires. They are disclaimed because they are objectionable. The validity of this mechanism cannot be doubted. In studying human behavior we see evidence of its dynamic force in every day life, in psychoneurotic as well as psychotic reactions. Fanatics on morality, severely critical of other people are not so infrequently found to have those tendencies or cravings which they despise in others. It was in 1911 that Freud stated the connection between projection and paranoia and was able to show this mechanism at work in individual cases. In Freud's analysis of Schreber's autobiography—he had not analyzed the patient himself—from memoirs which the patient had written following hospitalization because of a paranoid state, Freud pointed out as typical the association of paranoia with strong homosexual impulses. These the patient is unable to accept and therefore, copes with them through the mechanism of projection. In sweeping generalizations, the delusions and hallucinations of all paranoids were assumed to be projections. For the paranoid, who represses his homosexual impulses inadequately "I love him" becomes through the mechanism of reaction formation, "I hate him" and finally, "he hates me," thereby explaining the persecutory content of delusions and hallucinations.

Applying the mechanism of projection in a slightly different manner to the understanding of the delusion of marital infidelity, for example in the chronic alcoholic, Freud states: "The part played by alcohol in this disorder is thoroughly intelligible. We know that drink removes inhibitions and undoes the work of sublimation. It is not infrequently that disappointment over a woman drives a man to drink—which means, as a rule that he resorts to the public-house and to the company of men, who afford him the emotional satisfaction which he has failed to get from his wife at home. If now this man becomes the object of a strong libidinal cathexis in his unconscious, he will ward it off with the third kind of contradiction: 'It is not *I* who loves the man—*she* loves him,' and he suspects the woman in relation to all the men whom he himself is tempted to love."

To such a degree has projection been accepted as the basic mechanism in paranoid delusions by a great number of psychiatrists, that even statistical conclusions have been drawn on this basis concerning the personality structure of patients who present a certain content in their delusions and hallucinations. In this way Gardnel* found among 120 consecutive admissions diagnosed either as "paranoid dementia precox" or "paranoid condition" that in 45% homosexual trends were the bases of the delusions. Among the male paranoid dementia precox cases as a group alone the percentage was 61.

If the delusions or hallucinations expressed openly or through symbolism that the patient had been attacked in this way the above conclusion was drawn. Such deductions stand and fall with the validity of the assumption that projection is *the* mechanism responsible for paranoid delusions. This problem we investigated through studying the dynamics of a large number of paranoid reactions over a period of several years. In this paper we concentrate mainly on one type, the delusion of marital infidelity or as Freud and others have named it, the delusion of jealousy.

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*Gardner, G. E.: Evidences of Homosexuality. *Psychol. Rev.* 18: 1057, 1931.

Why in Freud's psychological interpretation of the paranoid reaction the mechanism of projection predominates to the exclusion of others may be understood from the fact that his dynamic considerations center around the complex of guilt or the "taboo" —the forbidden. This was obvious when he developed his concept of repression. The conflict arises when instinctual urges from the "Id" strive for gratification but are not acceptable to the ego under the control of the "superego." A feeling of guilt prevents realization of the "objectionable" mostly incestuous impulses in actions or thoughts and, therefore, they are repressed. In harmony with his theory of instincts Freud has restricted the conflict situation to the sphere of drive quests and opposing forces or in his own words "the conflict between sexuality and the ego as it crystallizes around the oedipus complex." The various mechanisms attempt to stave off the feeling of guilt. Undoubtedly guilt is a crucial dynamic factor in many neuroses especially the obsessions and phobias. However, making it *the* general principle does not do justice to the variety of human conflict. In fact the super-ego as a moral censor has little or no significance in the dynamics of some neuroses. The analysis of many cases reveals that the capacity to react with the feeling of guilt is poorly developed. Of equal importance as a source of conflict is the threat to the feeling of self-esteem—of self-value. Nietzsche had taken this dynamic factor in consideration when he recognized repression—although not using this term—as a general human attitude, describing it in a lucid picture: "I have done this—says my memory; I can't have done this—says my pride and remains implacable. Finally—the memory yields." Here we recognize pride in the place of conscience and, therefore, endangered self-esteem of which pride is an aspect in place of guilt. Somewhat kindred ideas were applied by Alfred Adler to the neuroses in stressing need for self-esteem as the dynamic factor for the abnormal exaggerated goal setting of the neurotic. Cognizance of the psychopathology of self-value is of greatest importance in many psychotherapeutic situations.

In the paranoid reactions a threat to the ego-consciousness prevails frequently and although we have found the mechanism of projection in a number of cases at the root of the reaction, more commonly a mechanism which we have named "transvaluation"* aids the paranoid in his struggle for self-assertion. We have chosen the word "transvaluation" because it expresses what actually happens. The paranoid changes his experiences in the environment to fit his needs. A reality which is unbearable to the individual's feeling of self-value is transformed. In its faintest forms it is a mechanism at the disposal of and made use of by everybody, no less important or frequent in the psychology of everyday life than repression.

Although repression and transvaluation are working in everybody to a certain extent, it is obvious that one or the other are predominant in certain individuals. Correlations between type of mechanism and type of character have been established to a limited degree only. The solution is a future problem for characterology. The following situation may illustrate the variety of possible reactions and mechanisms in different individuals: A young man fell in love. He believed and had reasons to believe his feelings were returned. His thoughts and plans for the future centered around his love, when he suddenly realizes that the affections of his beloved have turned to another man. Experience shows individually different reactions of average of pathological nature to the blow which the feeling of self-value has received. Primitive reactions of rage may lead to homicidal actions against the beloved or her lover. A panic of despair can lead to suicide. Others may go into a reactive depression or a psychogenic stupor. One person writes a volume of sonnets redeeming himself through sublimation. Finally we see the mechanisms of repression and transvaluation. We will dwell upon the latter only. Transvaluation takes the form of depreciation in this case. It becomes a

*The term transvaluation has been used by Freud, however with a different connotation. He refers to the "transvaluation of all psychic values" that takes place between the dream-material and the dream.

weapon in the struggle for self-assertion, when the ego is faced with the impossibility of realizing its aims. Depreciation of the beloved alleviates the defeat. "She does not love me" is replaced by "she is not worthy of my love." Thus aggressive tendencies help to overcome the humiliating experience through degrading the beloved. However, this requires motivation. Therefore, a certain meaning is read into her words and actions. Her glances toward other men now appear frivolous; her movements betray a great sensuality, a whisper is subject to suspicion, a blush is interpreted as guilt. Because he had previously seen her in company of other men, it is now established for the disappointed lover that she goes with anybody. So far the reaction does not reach a degree as to impose itself obviously in contrast to reality. Nevertheless, it differs from a delusion only in a quantitative way, it is a "normal" counterpart to the delusion of jealousy or marital infidelity. The mechanisms are the same, the tendency to depreciate and interpret in one direction with resistance to correction of emotional judgment.

In the common delusion of marital infidelity the purpose of depreciation expresses itself convincingly and often takes grotesque forms. A well-known example is the paranoid, chronic alcoholic who when finally rejected emotionally and physically by his wife accuses her of having not one lover only, she has sexual relations indiscriminately with every fellow that crosses her path. He accuses her of incest with brothers or her own son and every possible perversity. However, in the delusion of the chronic alcoholic also a physiological factor has often to be considered among the determinants of the delusion. We hear from him that his potency had decreased and he, himself offers an explanation as to the cause: He was too disgusted with the promiscuous behavior of his wife. As a matter of fact, chronic alcoholism decreases the potency but at the same time, at least in the delusional cases, I found this to be true—increases the desire. It is no coincidence that the delusion of marital infidelity is so common among alcoholics and *never* observed among addicts to morphine or other opiates. The discrepancy between decreased potency and increased desire is not present in these because opiates decrease both potency and desire.

The following case of delusion of marital infidelity which I would classify as belonging to the group of sensitive paranoia gives good insight into the structure and origin of the delusion. The couple, he a 59 year old man, she four years younger, were referred for treatment by their physician because the husband continued to threaten his wife more or less seriously to confess her unfaithfulness with several other men. Although the accusations started as far back as ten years ago, and although two years ago the husband had threatened her with a gun, the wife had only recently confided in her son-in-law and her physician because there was a secret involved which she wanted to and has kept from her daughter up to the present day. This daughter, now 37 years old and happily married in the East, was born on the day the couple were married, in fact, several hours before. The marriage ceremony was performed in the hospital soon after the birth and the understanding physician dated the time of birth a few hours later so that the daughter was born legitimately. When on a later occasion the daughter required a birth certificate, the couple changed the marriage date on the document ante-dating it one year. The son-in-law, whom I had also the opportunity to interview, knows the facts now and agrees with the parents that they should be kept from his wife, their daughter. The reason why the wife had finally sought help was the husband's attempt to get hold of their mutual savings and deposit them in the safe box which was in his name only. His motive: He rightly feared that his wife might leave him some day; two years ago after the gun threat she had left home secretly and had gone to her daughter for six weeks without, however, revealing to her daughter and her son-in-law the nature of her husband's accusations. This flight from home incidentally gave new nourishment to patient's delusions—his wife had surely traveled with a man—otherwise she would

have told him of her departure, etc. The way in which patient attempted to confirm his suspicions, observed his wife and interpreted insignificant incidents as support for his delusion did not differ from similar cases. He was convinced of her lascivious behavior, but he was willing to forgive and forget if she would only confess and reform. The history will give us an understanding of the underlying dynamics which made him reaction-sensitive to the birth of his daughter up to the point that it overshadowed his further life as a humiliating experience. Patient and his wife were born in a small town in the State of Indiana which in the meantime has developed into a fairly large city. They have lived there ever since. The patient was the oldest of five children. The father was a policeman and the family had to live on his salary of $50 a month. Conditions were very poor at home, enough food was provided but his clothing was such that he was ashamed to go to school. For example he had to wear his mother's old button shoes and the other children made fun of him. The attitude of the other children, who looked down on him as poor, made him sensitive at an early age, he feels. He was ambitious to get ahead in life and was a good student. However, he had to leave school after the 7th grade because at that time the parents were in danger of losing their home. So at the age of 13 years he went to work in a shirt factory. His weekly pay check of $3.60 was given to the mother who gave him ten cents weekly which he spent on candy. He attended night school and studied mechanical drawing. At 15 he earned $2.50 a day which was more than his father made. Up to the age of 20 years, he gave all his earnings to his mother, then he started to save for himself. He took violin lessons and invested his savings in a lot in a good residential section—he planned for the future. These were obviously the happiest years of his life; he played on the city baseball team and had dates with girls. Earlier he had felt inferior to other young people and was especially bashful toward girls; now he started to develop self-confidence. At 20 years he fell in love with his future wife—she was not quite 17. Soon they went steady and after 6 months they became intimate; this led to the pregnancy nine months before they were married. From the start her family was violently opposed to him. The reasons he gives are confirmed by his wife. Her family felt he was not good enough. His family was poor and had no prestige and her family had money inherited from a grandfather. Her family ridiculed his manner of dress and criticized his manners; his wife made him feel that he did not know about the finer things in life. He proposed but she said her mother would not let her marry him. In fact, later he was forbidden to enter her house and they met secretly. When she realized she was pregnant, she did not confide in him for many months. After she had told him, he suggested eloping but she refused. He wanted to go to her mother and insist on marriage but he never found the courage to do this. Finally they were married under the circumstances described above. At that time he was working in a factory as a toolmaker. When he returned to work after his marriage and the birth of his daughter, he became the target of coarse jokes by his fellow workers. He left the toolroom after a few days and took a job in the designing department of the same plant. Later he joined the fire department and was gradually promoted to the position of a captain. He was a good officer, strict but just and very exacting. One day he reprimanded a lieutenant because of behavior outside of his duty unfitting for an officer. The lieutenant retorted that patient's private life had not always been above reproach. Soon after this incident he resigned from the fire department, therewith burying his ambition to become chief of the city's several fire companies, a position which he feels he had a good chance of getting because of his record.

Soon after his marriage he had begun to withdraw from social contacts which he had made seven years earlier when his economic status improved. The circumstances of his marriage and the birth of his daughter became for this basically sensitive and insecure man, a blow to his pride from which he never recovered. The fact that he had lacked the courage to bring it to a showdown

with his wife's family at an earlier stage enhanced his feeling of humiliation.

Analysis of the patient's psychosexual development and behavior was aided also by information gathered in the interviews with his wife. It revealed him as a man of strong and sustained drive. His sexual demands were great and characterized by a trait of possessiveness. When after the first two years of married life his wife did not always respond to what she felt were excessive sexual demands, although she did not refuse him, he began to ruminate whether she really loved him. Would she have married him without being pregnant. Why had she not been willing to elope with him? Was it not her and her family's fault that everything came as it did? Was she and her family really so much better? He found fault with her; she became less attentive to him. This confirmed his belief that he had made all the sacrifices for a woman who did not really love him. Finally he degraded her by his accusations as the opportunity offered itself. This was the case when an employer brought her home in his car in the rain.

It is not difficult to see why in a state of abnormal emotional tension reality lends itself to a delusional transformation. Perception is a complex mental act and into every perception subjective elements normally enter. Perceptions of the same incident by various observers may be extremely different, as well as perceptions by the same observer at different times, in different moods, at different age periods. Memories have an even higher degree of subjectivity. In the course of time they can be transformed to such a degree that the individual himself is frequently in doubt how much of his memories is reality and how much fiction.

The meaning of every experience finally is determined entirely subjectively by negative or positive valuation. Criteria for the evaluation lie in the individual, and what experience in the environment amounts to, is colored by the reflection they have on him. The appreciative critic is highly thought of, while the adverse critic is esteemed incompetent. Any movement or ideology supporting the individual's ego and everything he stands for will be rated highly and vice versa. Achievements akin to his own, his followers, his group are greatly estimated. Tastes and opinions are molded accordingly and a world view is formed with regard to the individual's place in it. Among scientists we can occasionally observe that theories which have become fundamentally important to the individual's ego for reasons of self-respect are defended with fanaticism shutting out contrary facts from consideration and thereby conforming to the paranoid pattern. Constant negative or positive valuation responds to the needs of the ego. By thus transvaluating the meaning of experiences in the environment, the ego protects itself and copes with an adverse reality.

I come to the conclusion that a mechanism found very frequently in paranoid delusions is that of transvaluation, in which unconsciously emotionally demanded changes in the preceptional material, the memories and the meaning of experiences work together to transform reality so that it becomes compatible with the needs of the individual. We have said at the onset that other mechanism, for example, projection also rationalization or compensation may be at work in a number of cases. But transvaluation differs decidedly from projection through which, one attributes to others, one's own objectionable attitudes, motives and desires, or from rationalization through which we substitute fictitious motives for our true but objectionable motives. Compensation finally is a very comprehensive and not clearly specific mechanism by which we strongly develop some means of satisfaction in coping with anxiety resulting from frustration. Transvaluation is a mechanism as ubiquitous in our opinion as repression. We can make ourselves readily aware of it if we analyze our reactions in every-day living.

Recognition of the mechanism of transvaluation as important for the understanding of many paranoid delusions as well as the fact that the dynamics in these delusions differ should caution us to jump at foregone conclusions in the interpretation of delusions and also of hallucinations which usually are the concrete symbolic expression of delusional ideas. Every delusion requires an individual approach.

The Psychiatrist in Court

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The psychiatrist testifying in court is faced with the cold fact: On what will the court permit him to base his opinion? If the court is lenient it may permit him to base his opinion on any pertinent information that has come to his attention. Naturally this information is usually not obtained by the psychiatrist personally, but is obtained through associates—social worker, psychologist and correspondence. This last may be in the form of school records, hospital records, etc. Frequently this is in the form of a confidential and privileged communication which he is not permitted to introduce into the court record. The reports are usually not sworn to so in many instances they likewise are not admissible. Therefore, the psychiatrist must base his opinion mainly on the mental examination and observation of the prisoner. Naturally the prisoner is not going to give information that will be damaging to himself, if he can help it.

If the case is strongly contested the psychiatrist may find his testimony limited to what information he, himself, has obtained. I have had to give opinions without the aid of psychometrics, social histories or physical examinations because they were not admissible being considered hearsay evidence.

In 1942 in the trial of a general paretic who killed his father. I testified that he could not cooperate with his attorney and a jury returned a verdict of "insane." In 1949, without an examination by the Behavior Clinic psychiatrists, he was returned for trial on the murder charge and I was called upon to testify relative to his mental condition in 1942. The court, however, limited my testimony to his mental condition at the time of my examination, which was several months after the crime. I had not performed the blood test nor physical examination myself so I was not permitted to take them into account.

In Illinois, being found insane does not relieve the individual of responsibility for the crime so that upon complete and permanent recovery from his mental illness he must stand trial. Insanity postpones the trial but does not relieve responsibility. Of course, on the trial, the defense attempts, by means of expert testimony (i.e. that of the psychiatrist), to prove that the individual was insane at the time of the commission of the crime and therefore was not responsible. This expert opinion is brought out by the posing of a long hypothetical question.

The time that the individual is confined in the mental institution is not considered part of the sentence, although in some cases upon returning to court for trial the charges are dropped because of the hospitalization. We had an instance of a man who became insane in the County Jail after completing five months of a six month sentence on a conviction for committing malicious mischief. It was only after eight years in an institution for insane criminals that his transfer to a civil institution was effected.

If the psychiatrist is retained by the prosecution or the defense, he is going to base his opinion in large part on material furnished by the one that engages him. The only impartial witness is one who is selected by the court and who is able to take into consideration information obtained from all available sources. If the opinion is based on a strictly legal interpretation it may be strictly limited to the time of the examination, and then only by means of a hypothetical question, the mental state of a hypothetical person at the time of the crime may be discussed. Although the actual person is not named, everyone present knows that the mental state of the prisoner in court is under discussion.

The psychiatrist must limit his answers and elaborations to facts, circumstances and events set forth in that hypothetical question. He is not allowed to relate any addi-

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tional facts that he may know about the case. He is required to answer the question directly and not volunteer information. If the witness feels that the question cannot be answered in the form presented, he may request permission of the court for clarification of the questions but his answer remains confined to some form of legal question put by lawyers.

Recently I appeared as an expert witness in the trial of a 12-year old defendant indicted for murder. In the State's hypothetical question the actions of the boy were related, step by step, for hours before and after the crime. To this hypothetical question I answered that he knew the nature of the charge and was able to cooperate with his counsel—that he knew the difference between good and evil. The hypothetical question of the defense contained pages and pages relating to the boy's background. It brought the boy up to the date of the crime, at which time he was said to have been called an obscene name. He then "blacked out," and remembered nothing until hours later he was covering the body with leaves. The hypothetical question of the defense stopped there. To this question I answered that in my opinion the hypothetical individual did not know the difference between right and wrong (good and evil) at the time of the crime.

Naturally, each side wishes to introduce material into the hypothetical question to make the hypothetical question most favorable to its side. Extreme care is taken as to the language and phraseology. For example, in one case the testimony was that the individual had drunk 4/5 of a quart of whiskey. When the hypothetical question was read the attorney read it as "four or five quarts of whiskey," which the state's attorney did not notice nor object to.

Occasionally the court may ask us our opinion of the mental condition at the time of the crime, stating specifically that we may take into consideration any information obtained. In such instances the testimony of the arresting officers, observations by others in a position to know about the defendant and confessions are very important and may be taken into account.

In Cook County, to eliminate the so-called "battle of the alienists" the Behavior Clinic of the Criminal Court of Cook County was established in 1931. This was at the instigation of the Chicago Medical Society, the Institute of Medicine and the Chicago Neurological Society. The authority for the Clinic was established under an act of the legislature which gives the Bureau of Public Welfare the right of establishing needed medical services to counties over 500,000 in population. The Behavior Clinic examines prisoners only upon court order obtained upon the request of the State or defense. Under a ruling of the Illinois Supreme Court the presiding judge is not permitted to appoint a psychiatrist to testify for the Court, as in that case there would be a tendency for the jury to give more credence to the judge's psychiatrists than to those of the defense and prosecution. Occasionally the State may request an examination, but the defense may object, wishing to use its own psychiatrist. The State may then bring out the point it wishes to stress by means of a hypothetical question. If a defendant refuses to cooperate in the examination by the Behavior Clinic, on the advice of his attorney, it is not permitted to introduce such evidence into the trial. Each side may request the psychiatrist from the Behavior Clinic to testify, which means that the most poverty-stricken defendant may receive the same expert psychiatric attention as the wealthiest. The report of the Behavior Clinic has been accepted in most cases by both defense and prosecution as unbiased. Before the establishment of the Behavior Clinic it was customary for the defense and the State to hire the same number of alienists. If the defense, for instance, had the means to hire three, or even five, it was almost obligatory for the State to do likewise.

In Illinois one may be—

(1) insane at the time of the offense but sane at the time of going to trial;
(2) insane at the time of the offense and insane at the time of going to trial;
(3) he may become insane before going to trial and be insane at the time of going to trial;
(4) become insane during the trial;
(5) become insane before sentence;
(6) become insane before execution of sentence.

If at any time the question of sanity is brought before the court it is mandatory to impanel a jury of twelve to pass on the mental condition of the individual. In Illinois there are special tests to establish the responsibility of an individual at various stages of the trial. These are as follows:

At Commission of a Crime: When an accused is put on trial for a crime or misdemeanor, the test as to insanity is whether or not the defendant is capable of knowing right from wrong as to the particular act in question and is capable of exercising the power of choosing either to do or not to do the act and of governing his conduct in accordance with such choice.

Before Trial: He is not considered a lunatic or insane if he is capable of understanding the nature and object of the proceedings against him, and if he rightly comprehends his own condition in reference to such proceedings and has sufficient mind to conduct his defense in a rational or reasonable manner, although upon some other subjects his mind may be deranged or unsound.

Before Judgment: A person that becomes lunatic or insane after the commission of a crime or misdemeanor shall not be tried for the offense during the continuance of the lunacy or insanity. If, after the verdict of guilty, and before judgment is pronounced, such person becomes lunatic or insane, then no judgment shall be given while such lunacy or insanity shall continue. And if, after judgment and before execution of the sentence, such person becomes lunatic or insane, then in case the judgment be capital, the execution thereof shall be stayed until the recovery of said person from the insanity or lunacy. In all of these cases, it shall be the duty of the court to impanel a jury to try the question whether the accused be, at the time of impaneling, insane or lunatic.

Before Execution: The defendant before execution is to be regarded as sane and not lunatic, when he has sufficient intelligence to understand the nature of the proceedings against him, what he was tried for originally, the purpose of his punishment, the impending fate which awaits him, and a sufficient mind to know any facts which might exist which would make his punishment unjust or unlawful, and sufficient intelligence to convey such information to his attorney or the court. When he has not such intelligence and mental ability he is to be regarded as insane or lunatic by the verdict of the jury, if so found, and his execution stayed or prolonged.

The same tests apply for feeble-mindedness and insanity in Illinois, that is, one must be unable to distinguish between right and wrong and not be able to cooperate with counsel.

In relation to the responsibility of minors, the law of Illinois provides: "Infants under the age of ten years shall not be found guilty of any crime. They are considered of sound mind, if neither an idiot or lunatic, and who have arrived at the age of 14 years, or before that age if such person know the distinction between good and evil."

If a defendant before the court has ever been committed as feeble-minded or insane (mentally ill), a restoration must be decreed in order to prevent a reversal of the verdict and sentence at some future time. This restoration consists of a hearing before a jury of twelve.

A person is not criminally responsible for an offense if at the time of its commission he is so mentally unsound as to lack the knowledge that the act is wrong. That is the "right and wrong" test. This part of the rule prevails in all but two of our states. This is the sole test for avoidance of responsibility in England and in 29 American states. The wording of the rule, however, varies considerably; in the majority of jurisdictions it is stated that to be excused the defendant must be so disordered as not to know the "nature and quality" (sometimes "nature and character," "consequences," etc.) of the act, or if he did know what he was doing, that he did not know it was wrong.

In 1843 in Ohio a jury was instructed that if the defendant at the time of the act could discriminate between right and wrong and was conscious of the wrongfulness of the act and had the power to forbear or to do the act, he was responsible. This was the introduction of the "Irresistible Impulse Doctrine" into the American Courts.

In the following year in Massachusetts Justice Shaw charged a jury: "If it is proved, to the satisfaction of the jury, that the mind of the accused was in a diseased and unsound state, the question will be, whether the disease existed to so high a degree, that for the time being it overwhelmed the reason, conscience and judgment, and whether the prisoner in committing the homicide, acted from an irresistible and uncontrollable impulse: if so, then the act was not the act of a voluntary agent, but the in-

voluntary act of the body, without the concurrence of a mind directing it."

Since that time, in 17 states, will-power enough to resist the impulse to commit a crime has been added to the knowledge that the act is wrong.

To repeat, 29 states base criminal responsibility on the so-called "Right and Wrong Test": Seventeen states and the District of Columbia add the second test of "Irresistible Impulse." In these latter jurisdictions a person is excused if he is incapable of knowing the wrongfulness of the act, or even though he does know it is wrong, if he is incapable of controlling the impulse to commit the act.

In New Hampshire the court rejected both tests and formulated the rule that there is no legal test of irresponsibility by reason of insanity: "It is a question of fact for the jury in each case, whether the defendant had a mental disease, and if so whether it was of such a character or degree as to take away the capacity to form or entertain a criminal intent."

In Rhode Island the court has never passed upon the question of a legal test of insanity.

In Nebraska feeble-mindedness and mental disease have been held grounds for mitigating punishment.

In Illinois if the defendant is sentenced to the penitentiary a complete file of the Behavior Clinic consisting of our social history, mental examination, psychological examination and report to court is forwarded to the penitentiary. Our report to court consists of: (1) statement of the authority for the examination; (2) a summary of the physical examination; (3) a summary of the social history, not citing previous incarcerations; (4) a summary of the mental examination, not citing previous incarcerations, or the offense, or previous offenses; (5) a medical diagnosis; (6) whether the defendant knows the nature of the charge and is able to cooperate with his counsel. If he is unable to cooperate or his mental state is such that he does not know the nature of the charge, he is presumed to be insane, and it is mandatory to impanel a jury of twelve to pass on the mental condition of the defendant.

Often we are asked the question in court: Do we have an opinion as to whether the defendant is insane? If not objected to, the question is answered, but in that case we are invading the province of the jury.

Frequently claim of insanity is used by the defense to delay a trial, to wear out the witnesses, etc. A case may be ready to go to trial and then the defense brings before the court the issue of insanity. It is then mandatory for that issue to be disposed of before the trial can proceed. When the defense attorney can provide no other defense he can always claim that the defendant was temporarily insane and now has recovered, so that he would not have been responsible at the time of the commission of the crime. We had one patient who claimed to be insane after three o'clock in the afternoon, at which time the jail was closed so that we were not able to observe him in his so-called periods of insanity.

If the prisoner is facing the electric chair one must be suspicious of malingering. We had one patient, charged with murder, on whom we were about to report to court that he was unable to cooperate with his counsel. However, a letter he attempted to pass to his wife during a visit was intercepted by a jail guard, read in part: "I am not supposed to be aware of anything, as I am supposed to be crazy. I want to impart some information to you which you in turn will give to the lawyer representing me. Don't you realize that my life hangs in the balance, that everything depends on you, as I put my trust in you? So please, for God's sake, heed my words. Don't you realize that they want to make an example of me and put me in the electric chair, even though they may not be able to do it if I go to trial. It is too late to turn back now. I have gone too far and they are mad at me because if I am pronounced insane it will spoil the State's case and they will have to commit me to the Psychiatric Division until I am cured, and then I will come back and have a chance to beat the rap, because I can contend that I was not responsible for my actions. After you have copied the information burn this

letter. I want you to volunteer this information of your own accord because you naturally know all about my life." He then gave a list of friends to call upon to testify that he gave the impression of being "unstable, irrational and unsettled"; that he would always lose interest in things he was doing; he would begin a conversation and suddenly stop in the middle of it and start roaming; that he would sit and stare into space for long periods of time, and would pay no attention to those about him. He advised that all his friends be informed of this and then that they be subpoenaed to testify in court. In a 12-page letter he outlined the steps one by one. Confronted with this evidence his behavior changed immediately. He submitted to the mental examination and no overt signs of mental disease were elicited.

We have had individuals come to court acting in a very bizarre manner, and the jury would find them insane. As soon as they leave the court room, their behavior would be normal. One, charged with murder, would enter the court room in a dishevelled fashion, and during the court procedure would throw his shoes and stockings around the floor. As it was in winter time the stone floors were cold, and it was noted that he attempted to reach his stockings with his feet, in order not to touch the cold floor. His sister was an excellent witness in his defense, collapsing on the stand. Later it was learned that she had taken dramatic lessons to prepare her for her court appearance.

We have had many interesting cases. Fear plays a large role in many instances. I have seen a man drop dead after leaving the witness stand; another attempt to drive through a barred window before going to court, although his offense was a minor one and probation had been assured him. Some attempt suicide and are successful. Others become panicky, others mute, some combative, some have epileptic seizures, others faint or have heart attacks.

Our greatest difficulty is with defense lawyers in cases of psychoneurosis and psychopathic personalities. Anyone who shows the slightest deviation in behavior from normal, or who was retarded in school, is considered by his counsel as insane in order to avoid the consequences of his acts. If the charge is serious and the individual has previously been committed as feeble-minded or mentally ill, the defense tries to avoid the legal tests and attempts to have him committed civilly. This is especially true in the borderline mental defectives and veterans receiving compensation. One must remember that the I.Q. could be higher, but not lower.

In closing, it is my recommendation, for the improvement of forensic psychiatry, that a complete psychiatric evaluation be made on all youthful offenders, and special attempts made to rehabilitate this group. We will continue to have difficulty in differentiating between insanity, that is, irresponsibility in the legal sense, and mental illness in the medical sense.

We should strive to do away with the insanity issue at the time of trial, and have the individual tried for the offense; then use psychiatry as a means of disposal and not as a means of defense, thereby eliminating much of the unfavorable impression of court psychiatry in the layman's mind. Psychiatry is now used as a defense and the guilt or innocence is not proven. The innocent, if found committable, suffers along with the guilty and is incarcerated with the guilty. The layman feels that psychiatry can be bought at a price and we have found that in widely publicized newspaper cases well known psychiatrists have offered their services.

It is further urged that legislation be generally enacted to provide for a panel of psychiatrists to be appointed by the court and made available to both sides; that psychiatrists testifying in courts have access to all information from both sides, to avoid partiality, and that their fees be paid by the State. That the hypothetical question should be abolished, that the expert testimony of the psychiatrist be kept along medical lines so far as it is humanly possible to do so, and last but very important, that newspapers do not glamorize crime.

The Psychiatrist and the Cancer Patient

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It has seemed to us that the social responsibilities inherent in the practice of psychiatry are extending increasingly beyond the circumscribed role of the physician-patient relationship. One of the manifestations of this responsibility is in aiding our medical colleagues in the development of more effective methods of prophylaxis and therapy by clarifying the actual factors functioning in the psychology of the individual, thus enabling ourselves as physicians to work more objectively and, perhaps, helpfully as regards the patient.

In studying the psychological patterns existent in the individual patient and the effect of the physician upon these patterns the one most fraught with disturbing emotionalism is that present in the cancer patient. Typical of this pattern is the following, perhaps too common, anecdote:

A surgeon speaking, stated, "Yesterday a lovely woman whom I have known for some time came to see me with a history of intermittent spotting of about three months' duration. We put her on the examining table and found a big, fungating cervical carcinoma." He was asked what he told his patient, and replied, startled, "Why, I told her that she had a condition that looked pretty irritated and that she might have to go to a hospital for some treatments." When questioned as to why he had not told her the true condition, he became annoyed, argumentative and inconclusive, finally stated that, "It depends on the individual." We will not attempt to survey the ramifications that can be followed in this line of reasoning because the argument itself is useless and unproductive. Rather an outline of human psycho-dynamics is suggested.

The concept that the so-called "instinct of self-preservation" is a most basic ego function is important. Perhaps, however, many of us have not followed through with our clinical awareness that this self-preservatory drive is directed toward the strengthening and maintaining of the ego—i.e., the dignity of worth-whileness of the self even to the point that the physical body will be sacrificed to maintain the ego. Hence, it would appear that the therapy of the cancer-threatened individual could be most effectively maintained if the physician is oriented toward accepting the individual's ego-concept of himself as an adult or mature human being. It is quite true that each individual must be viewed as unique but, in doing this it is suggested that certain guide posts in the viewing be followed. The most important would seem to be an attempt to answer the question, "How does this individual best sustain his concept of himself as a mature being?"

Fromm-Reichmann, in a recent publication pointed out that an element present in many schizophrenic illnesses was an apparent struggle between the individual's concept of his maturity and the twisted drives that he was subjecting himself to. We find this a plausible and easily recognizable complex and, to apply it to some actual case-studies we present the following:

A 70 year old male physician rather suddenly developed symptoms of acute urinary retention. Competent urologic examination revealed an enlarged smooth prostate and supra-pubic prostatectomy was performed. At the time of operation, to adopt a pertinent quote "nothing was suspected" but the clinical pathologist returned a report, a few days later, of "adenocarcinoma which has, apparently, infiltrated the capsule. Classification: Grade One."

The patient had two sons, both of whom were practicing physicians. Automatically they reviewed the evidence and, just as automatically both decided to "conceal the evidence" through the mechanism of the carefully cultivated and widely used medical double-talk. In this atmosphere the cancer patient moved along but became increasingly querulous, demanding of many petty attentions and annoyingly egocentric; as metastatic symptoms made their appear-

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ance they were put off as "probably arthritis" or "gall stones." The family situation deteriorated until one son said to the other, "I can't understand Dad being so damn neurotic." At about this time it was decided to employ psychiatric services and, at a conference, it was suggested that the sons acquaint their physician-father with the accurate medical facts of his condition.

The suggestion was followed, and the atmosphere changed overnight. Never once, according to the history given did the patient mention his condition thereafter. It became apparent that he conceived of himself as the one individual who would conquer his disease. The condition terminated in death about a year later but the family atmosphere had no element of the fretful, querulous neuroticism that had marked it previously. In formulation, one of the sons later said, "in retrospect, I realize that we had placed father in an intolerable spot. From having been the trusted, accepted adult male head of the family, we had suddenly tried to reduce him to the position of infantile dependency, sort of patting him on the head with a 'there, there, little man, we know best for you' attitude. In other words we were rejecting him and, I suppose, were doing so mainly in order to protect ourselves."

In therapeutic psychiatry, a prime objective constantly maintained by the psychiatrist is, through the various accepted methods of therapy, to help the individual to face, deal with and accept the facts of the so-called "reality situation." This is generally accomplished through a more or less prolonged method in which the individual, through his capacity for transference, reenacts and comes to understand the meaning of his reaction patterns and then, through the increased facility of his own human mental dynamisms, handles and controls his action patterns without incapacitation of himself or his environment. Why then, do we as physicians when faced with the patient suffering with the "dread condition" suddenly reverse our fields, drop all the painstaking technical knowledge which we have so laboriously acquired and regress to a floundering, rejecting and improvised but dishonestly rationalized and sanctimoniously self-justified level of performance? It is a fact that we, and most other physicians, do. It appears to be a fact that this performance contributes little, if any, ease to the patient and it could be that it results in actual harm in that it continues to nourish the concept of the "dread disease."

To find some answer to this cultural fear an attempted careful analysis of the meaning behind the monosymptomatic body delusion of cancer in a short series of four recently encountered patients was made. The hypothesis was that if the thinking patterns of these patients were clarified then perhaps light would be cast upon the primitive attitude of people today toward malignancy.

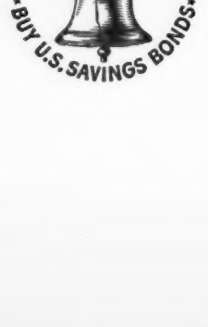
One patient, a 53 year old woman, referred for psychiatric treatment, came into the consultation room with a drawn, harried, angry look upon her face. She sat down and announced, "I have a cancer and I know it. I've been to 27 doctors in the last year trying to get someone to operate on me and none of them will. Several of them even had the nerve to say, 'Lady, you had better watch out, you are liable to find a doctor who will operate on you and then you will really be in trouble.' Well, I guess you'll be number 28." In taking the longitudinal history it developed that the patient was married to a hard-working uneducated farmer who took a submissive, placating attitude toward her. Her three daughters, all college graduates, were quite ambivalent in their feelings toward her. As one stated, "We know that this is just one of her ideas because ten years ago she had a goitre, at least she thought she did; but thank goodness nobody operated on her. But doctor, please be careful. If anything happens to mother we'll never get over it." Further questioning of the patient revealed a long, loosely organized paranoid reaction with extreme hostility directed toward her husband and children. She had introjected her feelings and, because she considered cancer as a "horrible eating disease that gnaws you to death" had visited upon herself a symbolic wish of her feelings toward her family.

A second case involved a woman who had developed an obsessive-compulsive illness in which she believed that "the germs of pyorrhea" could cause cancer and, therefore,

everything that she breathed upon had to be sterilized. She had introduced a unique twist in that while handling the bed clothes she would hold her breath until she was almost finished and would then "have to" explosively expel her breath and "the cancer germs" on the bedclothes. In a frenzy, she would remove the bedclothes and repeat the whole process until the laundry accumulated in a huge pile. Again, the intense hatred became evident. The mother, a rejecting, domineering person, had insisted to the patient that she would never be acceptable to anyone. The patient had met her husband in an office in which she worked to furnish the financial support for her mother. They became friendly and in the course of time she became illegitimately pregnant. Forced to marry, she found her husband to be an individual who insisted on sexual relationships two or three times a day up until she left for the hospital to have her baby, and he had then been annoyed because there would be no one at home to cook his meals. Her child had been born and she had become an over-solicitous mother only to have the child develop a badly-ailing heart due to rheumatic fever. The fact that she had focused upon cancer as her neurotic solution, again revealed the belief that it was "the most horrible thing that could ever happen to anyone."

Other cases have followed like patterns. The whole focus of the individual's hates and aggressions are focused on malignancy and the usual control methods and guilt-expiating mechanisms are exercised to keep the feeling-pattern in balance. Is it possible then, that the cultural view of malignancy likewise incorporates some of these aggressions and hostilities that exist in all of us? The very term malignant is used both as a medical term and as a term depicting malevolent, hostile and destructive attitudes. Pathologically, however, a carcinoma is lethal because of its invasive and usurpational powers, not because it "eats other parts of the body up." More classically, the bacterial diseases do this. However, cancer has taken on the role of the "bare-fanged monster which gnaws away cannibalistically" and perhaps because of this concept has become incorporated into the cultural patterns of today as the embodiment of the unspeakable, unacceptable and rejectable drives which, because of our cultivated awareness of them, we perhaps tend to reject more forcibly.

It is suggested then, that we, as psychiatrists, attempt through the educational means at hand to help ourselves and our medical colleagues to realize that the attitudes that we present toward the "Dread Disease" malignancy, are, possibly focused manifestations of our own unconscious attitudes and that, when we say, "I'm only trying to protect my patient from an intolerable awareness," it is because we are in reality trying to protect ourselves and our ego-structures from something which, in itself, is unreal and that, in so doing we are perpetuating a social neuroticism which in its blocking of many people from seeking early aid and its later emotional effects on the sick individual and his family epitomizes classical malignancy.



Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

Case No. NP 50-39

Presentation

A 45-year-old woman, a linotype operator, had been well until mid-November 1949, at which time a filminess appeared before her eyes. The blurred vision was more severe on the left. For several years previously there had been some mild tingling and numbness in the hands but she had paid decreasing attention to it and it never interfered with her occupation. Eighteen years previously a luetic infection was treated with bismuth and arsenicals.

Examination on November 27, 1949 revealed haziness of the optic disc margins with obliteration of the cups, and engorgement of the retinal vessels. There was no elevation of the discs. Deep reflexes were hyperactive. One week later a few hemorrhages in the discs and peripheral fundi were noted. A lumbar puncture revealed an initial pressure of 200 mm. (the patient was tense). The total protein content was 75 mg.%. The Wassermann reaction and gold curve were negative.

On December 31 she became restless. She talked less and her speech was thick. She complained of numbness in the left hand. In the course of the next few days progressive weakness of her left side developed. When examined on January 2, 1950 she had staggering gait, mild weakness of left extremities and moderate intention tremor on the left. The pupils were dilated and failed to react to light. Both optic discs were pale and well outlined. There was left facial weakness with preservation of ability to wrinkle the forehead. Her speech was thick. Deep reflexes in the legs were hyperactive with ankle clonus on the left. A left Hoffmann, and bilateral Babinski and Chaddock responses were elicited. No sensory disturbances (touch, pin prick, vibration, and position) were noted (including left hand).

She became totally blind on January 5. Ophthalmoscopic examination revealed questionable pallor of the disc on the right and a more obvious greyish pallor on the left. There was a proliferative reaction about each disc secondary to the previous hemorrhages. The pupils were widely dilated and did not react to light. There was a slight left facial paresis of the central type. Speech was slurred and scanning. Left hemiparesis with astereognosis was present. It was thought that some difficulty existed in identifying common objects by name. Deep reflexes in the arms were hypoactive. Abdominal skin reflexes were absent. Deep reflexes in the legs were hyperactive, and there was sustained ankle clonus.

Blood pressure was 110/86. Roentgen plates of the skull revealed hyperostosis frontalis interna. The pineal was not calcified. Cerebrospinal fluid protein was 82 mg.% and again the Wassermann and gold curve were normal.

From January 6 her course was progressively down hill. She was lethargic and dull. The hemiparesis increased in severity; the left arm became flaccid and left leg spastic. On January 9 her voice was hoarse and she had difficulty in swallowing. On January 12 she developed random searching movements; she would tap her mouth to indicate she wanted her false teeth reinserted. There was a frantic and perseverative type of behavior. She was completely aphasic. By January 14 she was deeply stuporous which progressed to coma from which she was never roused up to the time of her death on February 25, 1950. During this period she was incontinent of bowel and bladder, and had to be tube fed. Her pupils would be seen to be alternately dilated or miotic. Her extremities became rigid. There were episodes of respiratory distress with stridor and cyanosis relieved by oxygen.

Differential Diagnosis

Dr. Milton Rosenbaum: This 45-year-old woman obviously had a very acute and disastrous neurological illness. Apparently she

was well up to the middle of November. Her illness announced itself with visual disturbances—blurred vision orfilminess was complained of over her eyes. The original examination revealed haziness of the discs and obliteration of the cups and interestingly enough no focal neurological findings. At this point, quite correctly, lumbar puncture was performed, which revealed a pressure of 200 mm. of water, although there was a note that the patient was tense; the total protein was 75 mg.%.

Now if we put ourselves in the place of the physician who had to deal with the problem at that time, I think there is little doubt about what we would have had in mind. A patient with visual symptoms, with obliterated optic cups and haziness of disc margins, with an initial pressure of 200 mm. in the cerebrospinal fluid no doubt brings to mind brain tumor whether she was tense or not and the increased cerebrospinal fluid protein would be corroborative. In a situation such as this I think it's a good idea to keep brain tumor in mind; to keep thinking of the possibility of a brain tumor doesn't necessarily commit one to that diagnosis. There was one finding at that time which probably would make one a bit hesitant about this diagnosis and that was the nature of her visual disturbance. Here we get into a discussion of the differential diagnosis between optic neuritic and choked disc. I doubt if anybody who saw those discs could have stated that it was optic neuritis or early choking; the point that would favor optic neuritis is the fact that she complained of visual disturbances. The majority of patients with rather severe choking do not have visual complaints. Her visual symptoms would have made us suspicious of an optic neuritis as the cause of the visual disturbances rather than choking. There are a few things that are missing. There is no mention of visual fields, so I gather that visual fields were not done. Demonstration of a central scotoma would have settled this question.

At the same time syphilis is noted in the past history and naturally one wonders about central nervous system syphilis. The cerebrospinal fluid findings were against it, especially as the disease developed so rapidly and with such overwhelming signs. The blood Wassermann reaction was not mentioned; I assume that it was negative. Had the blood Wassermann been positive we would have been in more of a dilemma at the bedside. Vascular neurosyphilis has been known to occur in the presence of negative cerebrospinal fluid serological findings; with the serologically negative blood and cerebrospinal fluid (as well as the clinical picture) we are safe in ruling out neurosyphilis.

Then, there is the note about paresthesias that she had had for several years and to which she paid little attention, which, of course, immediately brings to mind the diagnosis of multiple sclerosis, particularly after witnessing the beginning of this catastrophic episode. I would still believe even with that part of the history in mind, that when she was first admitted one would have considered a brain tumor and would have kept the paresthesias in mind but wouldn't have been too influenced by them.

The examination of December 31 revealed a change in her condition. Within a few days she rapidly developed numbness of the left hand, weakness of the left side, staggering gait, tremor and then primary optic atrophy to say nothing of bilateral pyramidal tract signs. The onset of the optic atrophy certainly was most acute; not only were the visual symptoms acute but also the changes in the discs, some indication of how rapidly the latter may occur.

The left hemiparesis and staggering gait deserve a word. We are faced with interpreting whether the staggering gait was related to the left hemiparesis, or did it mean something else? I would be inclined to think that the staggering gait was not related to the hemiparesis. If you'll visualize patients that you've seen even with marked hemiparesis, they don't stagger. You might say that they dragged their foot or that they had a peculiar gait. The word staggering certainly makes us think of cerebellar involvement, probably the thick speech points in the same direction. Then, there is the tremor; the tremor, the speech, the staggering gait all imply that there are cerebellar lesions or involvement of cerebellar pathways. We are confronted with a pa-

tient who had involvement of several major systems in her central nervous system—the optic pathways, the pyramidal tracts and the cerebellar system. Also she had subjective sensory symptoms as well as motor signs.

In the next few days, she became almost completely aphasic. I think it's interesting that at first the dysphasic phenomena were interpreted as asteriognosis, although there was a note to the effect that perhaps the asteriognosis was only a manifestation of dysphasia. This aphasia became complete; so we see involvement of another portion of the central nervous system; then quite rapidly she developed signs of brain stem involvement with other speech and swallowing defects. One might wonder if the brain stem findings represented primary brain stem involvement or if they reflected supranuclear palsy inasmuch as we know that she had involvement of both sides of the hemisphere. She had bilateral pyramidal tract signs and she had aphasia.

As to differential diagnosis, the rapidity of course, and the fact that this was a diffuse process in the central nervous system (optic atrophy supervening rapidly) certainly rules out brain tumor. We have already ruled out syphilis. The diffuseness of distribution of lesions certainly fits the picture of multiple sclerosis, and I think that what we see here is an example of acute disseminated sclerosis.

A word might be said about Schilder's disease which is an acute demyelinating type of encephalopathy that attacks the white matter of the central nervous system on both sides. The few cases that I have seen have been in children, but Schilder's disease occurs as frequently in adults. I'm sure that cases that have been diagnosed as having Schilder's disease probably had acute multiple sclerosis—some indication of the similarity of clinical findings. In the differential diagnosis we may consider the age. As you know patients with multiple sclerosis ordinarily fall into the younger age group, but here is an example of a woman who was 45. We've seen patients here whose multiple sclerosis was manifested in the fifth and sometimes in the sixth decade. Multiple sclerosis is not always a disease of young people. Although Schilder's disease is fairly frequent in children, which multiple sclerosis isn't, it is also a disease of adults. In children Schilder's disease starts out with rather diffuse mental and neurological symptoms rather than with specific focal neurological signs. However, interestingly enough in adults, Schilder's disease quite frequently starts out with visual symptoms usually related to the occipital lobes. A point in the differential diagnosis is that cerebellar symptoms are not as frequent in Schilder's disease as in multiple sclerosis. In this case, certainly the cerebellar symptoms were quite prominent. The psychic symptoms in multiple sclerosis are usually in the nature of euphoria. We can't take time at this point to discuss the etiology of the psychic symptoms that are sometimes seen in multiple sclerosis, but most of us feel that they aren't specific in terms of the lesions and that in many instances they represent the total reaction of the personality to a chronic disease. There wasn't time for our patient to develop the usual psychological defenses against disability. In Schilder's disease the psychic symptoms, so-called, are those of overwhelming organic dementia. In Schilder's disease the course may be acute like this, usually there are no remissions and exacerbations. It either runs an acute course with death or a chronic course, while in multiple sclerosis, as you know, there are in many instances remissions, and I think that the fact that this woman had paresthesias some two or three years ago would help make the diagnosis of multiple sclerosis. Therefore, my diagnosis would be acute disseminated sclerosis.

Dr. Charles D. Aring: The students are in essential agreement with Doctor Rosenbaum. Most of them felt that this was a type of multiple sclerosis unusual in its rapidity. Many of them mentioned Devic's neuromyelitis optica, which we have come to regard as a synonym for disseminated sclerosis, and a few discussed Schilder's disease in much the same vein as Doctor Rosenbaum.

Pathological Discussion

Dr. Murton Shaver: Autopsy in this case was performed in another hospital. The

brain weighing 1260 grams was submitted after fixation in formalin, together with a portion of spinal cord.

On superficial examination the cerebral hemispheres were found to be approximately equal in size and symmetrical with normal convolutional pattern. The overlying leptomeninges presented slight opalescence but were otherwise unremarkable. There was some widening of the sulci in the frontal regions bilaterally. There was no evidence of hippocampel herniation or cerebellar pressure cone, the midline structures showed no evidence of displacement.

Coronal sections made through the cerebrum, brain stem and cerebellum revealed a normal ventricular system lined by grossly normal ependyma, and the tissue generally was fairly well fixed. Diffuse, irregular grayish areas were found scattered throughout the white matter and also in the right basal ganglia, where the white matter had a yellowish or pinkish discoloration. No focal areas of softening were seen. These areas of mottled discoloration measured up to several cm. in width in the centrum semiovale and appeared to extend into the cortex in some areas. Similar areas of discoloration were found in the pyramidal region bilaterally in the medulla, and in somewhat circumscribed area in the pons and the white matter of the cerebellar hemispheres.

The segment of spinal cord with overlying dura was not remarkable on superficial examination and serial sections revealed a small patch of yellowish-gray discoloration here and there, mainly in the white matter and in an anterior horn at one level of the gray matter.

Representative sections of the brain were stained grossly with Le Measurier's stain. These demonstrated areas of demyelinization in the optic chiasm, and diffuse irregular patches of similar demyelinization were found in the centrum semiovale bilaterally and in the basal ganglia, particularly in regions near the ventricles. These areas extended into the gray matter of the cortex in some cases. This stain applied to sections of the brain stem showed patches of demyelinization in the pons, while in the medulla the pyramidal tracts showed symmetrical bilateral demyelinization. Other areas of demyelinization were found in the white matter of the cerebellar hemisphere. Spots of demyelinization in the spinal cord were minute, but appeared to involve the anterior horn with the adjacent white matter of one side.

Blocks of tissue were removed for microscopic examination from several areas of the cerebral cortex, from basal ganglia, pons, medulla, cerebellum and several levels of the spinal cord. All were stained hematoxylin and eosin, phosphotungstic acid, cresyl violet, Loyez and Bielschowsky methods.

Sections from the cerebral cortex and basal ganglia showed patchy areas of demyelinization of the white matter, the myelin being almost completely replaced by clusters of large histocytes containing granular material. Phosphotungstic acid stain failed to demonstrate the presence of any glial fibrosis and only a few proliferating astrocytes were seen. Bodian stain demonstrated the preservation of occasional myelin sheaths near the periphery of the lesion, but here the sheaths were vacuolated, swollen and beaded. Throughout the major portion of the lesion fragments of myelin material were found with no preservation of the sheaths. Bielschowsky stain demonstrated marked swelling and destruction of the neurofibrils throughout these lesions. There was no inflammatory cell infiltration in any of these lesions and polymorphonuclears were seen rarely.

Sections through basal ganglia revealed widespread areas of this type of demyelinization involving the internal capsule associated with slight glial proliferation. There was no inflammatory infiltration. Similar areas were found in the pons. In the medulla similar demyelinization was found to involve the pyramidal tract bilaterally. These again were associated with only slight proliferation, a few gemistocytic astrocytes being found. The myelin sheaths were slightly better preserved in this latter area, but showed marked swelling and fragmentation for the most part. The tissue generally showed a spongiose appearance with sheets of large histocytes containing granules replacing the normal strucutres. Similar, but somewhat earlier lesions with

slightly more sparing of the myelin sheaths and less infiltration by histocytes were seen in the white matter of the cerebellum and in the region of the anterior horn on one side of the upper dorsal spinal cord.

Still earlier lesions were found, unassociated with any cellular proliferation, and these showed only vacuolization and rarefaction of white matter. They were found scattered in various portions of the white matter of the cerebrum, brain stem and cerebellum. They were not definitely related to any vascular distribution, although in some cases they were situated around a vessel.

The nerve cells were generally fairly well preserved in the cerebral cortex, although occasional focal areas of loss were noted. Some chromatolysis was seen in the cells adjacent to areas of rarefaction, while in the spinal cord anterior horn cells were markedly diminished in number in one area adjacent to a site of rarefaction.

Sections through the optic chiasm showed it to be almost completely replaced by a spongiose type of tissue containing large numbers of histocytes with granular cytoplasm. These cells were clustered around some of the blood vessels while myelin sheaths and fibrils were unrecognizable for the most part throughout the sections. There was no inflammatory infiltration here and no evidence of reactive gliosis, aside from occasional swollen astrocytes scattered throughout the tissue. The fibroblasts generally were somewhat increased in numbers.

Clinical Diagnosis

Acute disseminated sclerosis.

Dr. Rosenbaum's Diagnosis

Acute disseminated sclerosis.

Anatomical Diagnosis

Acute disseminated sclerosis.

Dr. Clifford McIntyre: Could you relate any of the lesions to perivascular areas?

Dr. Shaver: Only a few of them.

Dr. McIntyre: They didn't resemble acute disseminated encephalomyelitis?

Dr. Shaver: No.

Dr. Norman Chivers: Is the term Devic's disease now used synonymously with acute encephalomyelitis or should it be restricted to transverse myelitis combined with optic neuritis. In other words, is it now common practice to refer to any form of acute multiple sclerosis as Devic's disease?

Dr. Aring: I believe so. It has been pointed out that neuromyelitis is an inadequate term since in any case the lesions have been found to be more widespread than the optic tracts and spinal cord. All of these persons coming to autopsy have had cerebral lesions as well. I prefer the term acute multiple sclerosis or as the English call it, acute disseminated sclerosis.

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